who suffered from gastric intolerance, spitting of mucus and blood, profound depression, suppressed menstruation, cachexia and a tumor in the epigastrium. The tumor could be followed from the ensiform cartilage to the umbilicus, and from there, for about an inch to the left. It was cylindrical and pulsated. At subsequent examinations it was apparently a rounded mass and limited to the epigastrium, and at another time it was found only at the umbilicus. Under improved alimentation it disappeared a week before death. Autopsy showed no gross disease of the abdominal organs and the aorta was of the ordinary calibre. Dr. Post surmises that in these cases a relaxation occurs in the walls of the aorta, similar to that found in the skin and subcutaneous tissue. A. F.

THE GANGLION CELLS IN ACUTE MYELITIS.

Since the publication of Friedmann's article on "Degenerative Changes in the Ganglion Cells in Acute Myelitis," Dr. Karl Schaffer, at the Histological Laboratory of Budapest, has been making a number of microscopical studies on this class of cases. The joint conclusions of these two observers were as follows: (1) The pathological alteration of the ganglion cells was found to begin in a limited portion of the cell structure; if in the centre (homogeneous swelling), the periphery remained normal in appearance; if on the margin of the protoplasm (sclerosis) the centre of the cell was usually found intact. (2) The greatest resistance to the pathological process was found to be in the nucleus, and more particularly in the nucleolus, the tendency of affections of the nerve cells being to attack the periphery first, and then through a centripetal course the nucleus and nucleolus. (3) From the above results, the so-called partial degeneration of the nerve cells was found to be the physiological consequence of a diminished functional capacity of the cell. (Neurologisches Centralblatt, April 15th.) B. M.

MULTIPLE NEURITIS.

Dr. J. Pal, in an article on this subject, gives an exhaustive review of the etiology and pathology of this much-discussed disease. His final conclusions are based upon a careful research of all the data bearing upon the question and upon a rather large personal experience. It has been the author's endeavor in this paper to elucidate the following statements:

(1) That multiple neuritis is an independent affection of the peripheral nerves, not associated primarily with disease of the spinal cord. (2) That in the course of a multiple neuritis the central apparatus may become affected secondarily, through noxious material circulating in the blood. (3) That a connection may be established between the peripheral neuritis and the spinal centre through the various conducting paths. (4) That in the so-called neuritis of tuberculosis the symptoms of œdema and reduced faradic irritability are frequently absent.

The important question in the study of multiple neuritis has always been, What is its relation to the anatomical alteration in the spinal cord, which is usually found in these cases? The author thought that the hypothesis of Erb, that multiple neuritis was a secondary degeneration consequent upon disease of the ganglion cells of the anterior horns, was not proven. A deduction from the author's observation leads to the conclusion that the fibres of the anterior roots are not in direct trophic relation with the ganglion cells of the anterior horns, as it is generally accepted. Those cases of multiple neuritis, in which autopsy was performed, microscopical examination of sections of the cord revealed degenerative changes in the posterior columns, especially in Goll's column. Such alteration in the white substance was interesting from the probability of the possible relation of this disease to tâbes dorsalis. These changes have been previously described by other observers as being found in toxic diseases. Tuczek found after a most careful research that by far the most vulnerable portions of the cord were the posterior columns. Leyden and Oppenheim believed in the peripheral origin of tâbes. In one of the author's cases of polyneuritis there had been, besides various degenerative changes in the spinal cord, a tâbes cervicalis, these alterations being no doubt due to some toxic affection. It was further stated that not alone was the posterior column found diseased in some cases, but that any of them might become involved in degenerative processes, as such changes had been found in all the columns. The author thought it would appear from these facts that this condition of the nerve fibres did not indicate a systemic, but a localized disease. Further research has, however, led to the conviction that disease of the cord, in a case of polyneuritis, is undoubtedly brought about, as a secondary consequence of the disease of the peripheral nerves, through the conducting paths. These conditions of the cord are known to exist, but to accept them as originating from a spontaneous systemic toxic affection, is to pin oneself to a statement that has not as yet sufficient proof. In regard to the hyaline degeneration, these changes were found not due to multiple neuritis, but secondary to blood changes. In conclusion the author stated that from his experience the present classified symptoms of latent neuritis were misleading. He had in several cases of tuberculosis, where no œdema had been present, made examination of the nerves of the feet and found them degenerated. Two of these cases were interesting from the fact that in the course of the tuberculosis there had been no evidence of neuritis present, when œdema in the ankle joint appeared, followed the next day by peroneal paresis. Post-mortem examination of the nerves showed parenchymatous degenerative changes in the cutaneous branches of the dorsum of the feet, and also in the peroneal trunks. Again the condition of so-called cachectic œdema was brought about by a degenerative disease, one which was likely to produce a corresponding disease in the nerves. As to the faradic irritability being diminished or lost, the author found it almost normal in two of his cases. (Wiener klin. Wochenschrift.) B. M.

HYSTERICAL FEVER.

Dr. Boulay, in the "Medical Journal of Lille," has recently given us an interesting *résumé* of this strange and, one might add, doubtful manifestation of functional nervous disease. Although the power of hysteria to simulate all kinds of organic affections is well known, it might be supposed that this similarity must stop at such a symptom as fever, and yet the number of well-authenticated cases already on record makes it impossible to deny that this form of fever does actually exist, apart from any organic lesion whatsoever.

It should be stated that by fever is meant simply the elevation of temperature, the quickness of the pulse and the dyspnœa; the other symptoms, as the disturbances of the nervous system, the digestion, etc., being due, not to the fever, but to specific infection which is causing the disease.

It has been objected, that there are numerous causes of error which may account for the supposed fever in these cases. Thus a local vaso-motor disturbance might produce a local rise of temperature without any general febrile condition being present, or the patient herself might succeed in making the thermometer read higher than it should, in